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Influence of herbicides on Rhizoctonia root and hypocotyl rot of soybean

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Abstract

Rhizoctonia root and hypocotyl rot, caused by *Rhizoctonia solani*, is a common disease of soybean. Field studies established to observe how preemergence and postemergence herbicides affect the severity of Rhizoctonia root and hypocotyl rot on several soybean cultivars were conducted in Champaign, Dekalb, Monmouth, and Urbana, IL. Herbicides did not significantly ($P \leqslant 0.05$) decrease the *R. solani* disease severity index (DSI) compared to the control, but did cause some increases in DSI compared to the control at a low frequency in some years. In greenhouse studies, dimethenamid + metribuzin, pendimethalin, acifluorfen, and imazethapyr caused an increased Rhizoctonia root and hypocotyl rot severity compared to the no-herbicide control.

Keywords: *Glycine max*; Interaction; Plant disease

1. Introduction

The influence of herbicides on plant diseases has been studied on many different crops and pathogens. Several reviews in the last 30 years have cited cases where herbicides have either increased or decreased plant diseases (Bollen, 1961; Katan and Eshel, 1973; Altman and Campbell, 1977; Rodriguez-Kabana and Curl, 1980). According to Katan and Eshel (1973), four mechanisms can cause an increase in disease as the herbicide may directly influence pathogen growth, virulence of the pathogen, host susceptibility, and/or change the relationships between other pathogens and other organisms.

Rhizoctonia root and hypocotyl rot, caused by *Rhizoctonia solani* Kühn [teleomorph: *Thanatephorus cucumeris* (Frank) Donk], is a common disease of soybean [*Glycine max* (L.) Merr.] in the North Central United States (Doupnik, 1993). Rizvi and Yang (1996) reported that 27% of the fungal taxa isolated from soybean seedlings in Iowa in 1993 and 1994 was *R. solani*. The fungus may cause preemergence (PRE) and postemergence (POST) damping-off in addition to rotting of the hypocotyl and roots. Reddish brown lesions on the hypocotyl at the soil line are the typical symptoms of Rhizoctonia hypocotyl rot (Yang, 1999). Wrather et al. (1997) estimated a 108,000 tonne soybean yield loss in the top 10 soybean producing countries and a 68,000 tonne yield loss in the United States caused by Rhizoctonia and Pythium root rots combined in 1994. *R. solani* can cause up to a 48% yield reduction in small plots (Tachibana et al., 1971). The recommended disease management strategy is to use good cultural practices that promote seedling health during the early growth stages (Yang, 1999).

A few researchers have observed how herbicides can interact with Rhizoctonia root and hypocotyl rot of soybean. Bowman and Sinclair (1989) reported that soybean seedling vigor was decreased in *R. solani* infested soil treated with the herbicides alachlor, choramben, dinoseb, fluchloralin, or naptalam compared to seedlings growing in soil not treated with a herbicide in the greenhouse. Conversely, Bauske and Kirby (1992) reported that soybean growing in soil

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treated with several different dinitroaniline herbicides did not have increased severity of root and hypocotyl rot caused by *R. solani* in field and greenhouse conditions, compared with untreated soil.

There have been no reports on how POST applied herbicides affect Rhizoctonia root and hypocotyl rot of soybean. Previous reports of how PRE applied herbicides affect Rhizoctonia root and hypocotyl rot of soybean were studied on older cultivars, which may not be as tolerant to herbicides as newer cultivars. The objective of this research was to determine the effects of both PRE and POST applied herbicides on Rhizoctonia root and hypocotyl rot of common current soybean cultivars under field and greenhouse environments.

2. Materials and methods

2.1. Field study 1

Field study 1 was conducted at Champaign, IL and Urbana, IL in 1998 and 1999. At the Champaign location, two soil types, a Drummer silty clay loam (fine-silty, mixed, mesic Typic Haplaquolls) and a Flanagan silt loam (fine, montmorillonitic, mesic Aquic Argiudolls), occurred. At the Urbana location, an Elburn silt loam (fine-silty, mixed, mesic Aquic Argiudolls) and a Thorp silt loam (fine-silty, mixed, mesic Argiaquic Argialbolls) occurred. The same site was used both years at Champaign and was cropped to soybean in 1997. Different sites were used at Urbana each year and were cropped to corn (*Zea mays* L.) in the previous growing seasons. The soybean cultivars Asgrow 3704, Asgrow 3904, Jack, Pioneer 9362, Pioneer 9363, and Savoy were planted 19 May 1998 and 20 May 1999 at Champaign, and 17 May 1998 and 20 May 1999 at Urbana. The cultivar Pioneer 9362 was replaced with Pioneer 93B65 in 1999 due to unavailability of seed. Plots were planted 8 rows wide on 0.76 m centers, 7.3 m long, and later trimmed to 6.1 m long.

There were five weed management treatments. They consisted of a no-herbicide hand-weeded control, PRE applied pendimethalin (Prowl, BASF Corp., Research Triangle Park, NC) at 1.39 kg a.i./ha, PRE applied tank-mixture of dimethenamid (Frontier, BASF Corp.) and metribuzin (Sencor, Bayer Corp., Kansas City, MO) at 1.47 and 0.42 kg a.i./ha, respectively, POST applied acifluorfen (Blazer, BASF Corp.) at 0.42 kg a.i./ha + 1% v/v crop oil concentrate (COC), and POST applied imazethapyr (Pursuit, BASF Corp.) at 0.07 kg a.i./ha + 1% v/v COC. Preemergence herbicides were applied after planting, but before soybean plants emerged, and POST herbicides were applied approximately at growth stage V4 (Fehr et al., 1971). All herbicides were applied with a CO_2^- pressurized hand sprayer with 8003 flat-fan nozzles (Spraying Systems Co., Wheaton, IL) calibrated to deliver 187 l/ha at 207 kPa pressure. Weeds that emerged after all herbicides were applied, were removed by hand in all plots.

Ten random plants were dug with a shovel and collected from each plot 14 days after POST herbicides were applied. Roots and hypocotyls of each plant were evaluated together and given a single rating for severity of Rhizoctonia root and hypocotyl rot using a 0–5 scale developed by Cardoso and Echandi (1987) where 0 = no lesions, 1 = lesions < 2.5 mm, 2 = lesions 2.5–5 mm, 3 = lesions > 5 mm, 4 = lesions girdling plant and wilting visible on leaves, and 5 = seedling damped-off or dead. A disease severity index (DSI) was calculated for each plot by (percentage incidence × mean severity of 10 plants)/5.

The statistical design was a 6×5 factorial where the factors were cultivars and weed management treatments, respectively. Plots were arranged in a randomized complete block design (RCBD) with four replications. Locations and years were analyzed separately. Analysis of variance (ANOVA) was calculated using the general linear models procedure (PROC GLM) in SAS (SAS Institute, Inc., Cary, NC). Fisher's protected least significant difference (LSD) ($\alpha = 0.05$) was used to compare means.

2.2. Field study 2

Field study 2 was conducted at Monmouth, IL in 1999 and 2000. Different sites were used each year with corn being the previous crop each year. The soil type in 1999 was a Muscatine silt loam (fine-silty, mixed, mesic Aquic Hapludolls), and the soil type in 2000 was a Sable silt loam (fine-silty, mixed mesic Typic Hapludolls). The soybean cultivars Asgrow 3002, Pioneer 93B01, Pioneer 9363, and Siebens 2701 were planted 24 May 1999 and 15 May 2000. All cultivars were tolerant to glyphosate (Roundup Ready, Monsanto Company, St. Louis, MO). Plots were planted 4 rows wide on 0.76 m centers, 4.6 m long, and later trimmed to 3.4 m long.

Glyphosate (Roundup Ultra, Monsanto Company) was applied over the entire experiment for weed control at 1.12 kg a.i./ha prior to applying the herbicide treatments. The herbicide treatments consisted of PRE applied pendimethalin, POST applied acifluorfen and imazethapyr, and a glyphosate-only control. Herbicide rates were the same as those described for Field study 1. Herbicides were applied with a tractor-mounted sprayer with 8003 flat-fan nozzles calibrated to deliver 187 l/ha at 207 kPa pressure. Ten plants from each plot were collected and rated for Rhizoctonia root and hypocotyl rot using the same methods as described for Field study 1.

The statistical design was a 4×4 factorial, with factors being cultivars and herbicide treatments. Plots were arranged in a RCBD with four replications. The ANOVA was calculated using PROC GLM in SAS. Years were analyzed separately if there was a significant ($P \leqslant 0.05$) year by treatment interaction. Fisher's protected LSD ($\alpha = 0.05$) was used to compare main effect means; however, if there was a significant ($P \leqslant 0.05$) cultivar by herbicide interaction, then least square means were compared using the PDIFF option in SAS and were considered different when $P \leqslant 0.05$.

2.3. Field study 3

Field study 3 was conducted at Dekalb, IL in 1999 and 2000. The same site was used each year and was cropped to soybean in 1998. The soil types present were a Drummer silty clay loam (fine-silty, mixed, mesic Typic Haplaquolls) and a Flanagan silt loam (fine, montmorillonitic, mesic Aquic Agriudolls). The soybean cultivar Dekalb CX 285, which is glyphosate tolerant, was the only cultivar used and was planted 4 May 1999 and 8 May 2000. Glyphosate was applied across the entire field experiment at 1.12 kg a.i./ha for weed control prior to spraying the herbicide treatments. The herbicide treatments consisted of POST applied acifluorfen and imazethapyr, and a glyphosate-only control. Herbicides were applied at the same rates as in Champaign and Urbana locations with a tractor-mounted sprayer with 8003 flat-fan nozzles (Spraying Systems Co.) calibrated to deliver 187 l/ha at 207 kPa. Ten plants from each plot were collected and rated for severity of Rhizoctonia root and hypocotyl rot using the same methods described for Field study 1.

The experimental design was a RCBD with eight replications. Each plot was 4 rows wide (0.76 m row spacing) and 7.4 m long. Data were analyzed using the general linear models procedure (PROC GLM) in SAS (SAS Institute Inc.). Years were analyzed separately. Means were compared using Fisher's protected LSD ($\alpha = 0.05$).

2.4. Greenhouse PRE herbicide study

A greenhouse study was conducted using PRE applied herbicides. The study included two soybean cultivars, three PRE herbicide treatments, and *R. solani* inoculated and non-inoculated plants. Three seeds of either soybean cultivars Jack or Savoy were planted into 1000 cm^3 polypropylene pots containing a 2:1 sand:soil mixture. Pots were placed on a greenhouse bench and grown under a 16 h photoperiod. The photosynthetically active radiation (PAR) was measured to be 434 $\mu E/(m^2 s)$ (LI-170 Quantum/Radiometer/Photometer, Lambda Instrument Corp., Lincoln, NE), and the temperature was $26 \pm 3°C$. Pots were watered to saturation after planting, and twice daily thereafter. After emergence, plants were thinned to one plant per pot. Herbicide treatments in the PRE applied herbicide study were a no-herbicide control, pendimethalin at 1.39 kg a.i./ha, and a mixture of dimethenamid and metribuzin at 0.74 and 0.21 kg a.i./ha, respectively. Herbicides were applied directly to the soil immediately after planting in an automated spray chamber with an 80015EVS flat-fan nozzle (Spraying Systems Co.) calibrated to deliver 187 l/ha.

R. solani isolate 65L-2 (ATCC 66489) (AG 2-2), originally isolated from soybean in Illinois (Liu and Sinclair, 1991), was stored on 1.5% water agar at $-5°C$. To initiate inoculum production, a 5 mm-diameter plug of the *R. solani* culture was transferred to a 9 cm-diameter petri dish containing potato dextrose agar (PDA, Difco Laboratories, Detroit, MI) and placed inside an incubator at 25°C with 12 h light/dark cycles. After 2 days, a 5 mm-diameter plug was taken from the edge of the growing colony hyphal tip and transferred to a 9 cm-diameter petri dish containing PDA. The plates were grown in an incubator at 25°C with 12 h light/dark cycles. After 5 days of growth, the agar and fungal cultures from five petri dishes were macerated in 1 l distilled water for 1 min using a Waring commercial blender (Waring Products Corporation, New York, NY). The mycelial suspension was adjusted to contain $\approx 1.53 \times 10^4$ colony forming units (cfu) per milliliter. When soybean plants were at growth stage VE, two holes ≈ 0.5 cm in diameter and 2 cm deep were made in the soil around each soybean hypocotyl with a wooden rod. A syringe was then used to inoculate each hypocotyl by applying 2 ml of the mycelial suspension on and around the hypocotyl using a slightly modified method of that previously described by Wrona et al. (1981). The holes in the soil around the hypocotyl allowed the mycelial suspension to flow down to the roots. Plants were removed from pots 2 weeks after inoculation and soil was removed from the plant roots with running tap water, and roots and hypocotyls were rated for Rhizoctonia root and hypocotyl rot using a 0–5 scale.

The experiment was a $2 \times 2 \times 3$ factorial where the factors were *R. solani* inoculation, cultivars, and herbicides, respectively. The experimental design was a completely randomized design (CRD) with three replications. The experiment was repeated once over time in another trial using the same methods. Data from the two trials were pooled and analyzed together unless there was a significant ($P \leqslant 0.05$) trial by treatment interaction. The ANOVA was determined using PROC GLM in SAS, and main effect means were compared using Fisher's LSD ($\alpha = 0.05$); however, if there was a significant ($P \leqslant 0.05$) interaction among factors, then least-square means were compared using the PDIFF option in SAS and were considered different when $P \leqslant 0.05$.

2.5. Greenhouse POST herbicide study

A greenhouse study was conducted to examine the effects of POST applied herbicides. The cultivars Jack and Savoy were used in the POST herbicide study with the methods described above. Plants were inoculated with *R. solani* at the V1 growth stage using the methods described for the PRE herbicide study. Greenhouse conditions were the same as described for the PRE herbicide study.

There were five herbicide treatments, including a no-herbicide control, acifluorfen at a $1x$ and at a $2x$ rate ($x = 0.42$ kg a.i./ha), and imazethapyr at a $1x$ and at a $2x$ rate ($x = 0.07$ kg a.i./ha). Postemergence herbicides were applied with 1% v/v COC to soybean plants at the V2 growth stage using the automated spray chamber described above. Plants were removed from pots 2 weeks after inoculation with *R. solani*. Disease ratings were done using the methods described for the PRE herbicide study.

The study was a $2 \times 2 \times 5$ factorial where the factors were *R. solani* inoculation, cultivars, and herbicides, respectively. The experimental design was a CRD with three replications. The study was repeated over time in another trial using the same methods. Data from each trial were analyzed together if there was not a significant ($P \leqslant 0.05$) trial by treatment interaction. The ANOVA was determined using PROC GLM in SAS. Means of main effects were compared using Fisher's LSD ($\alpha = 0.05$); however, if there was a significant ($P \leqslant 0.05$) interaction among treatments, then least-square means were compared using PDIFF in SAS and were considered different if $P \leqslant 0.05$.

3. Results

3.1. Field study 1

Neither weed management treatments nor cultivars had an effect on Rhizoctonia root and hypocotyl rot severity at Champaign in 1998 (data not shown). Mean DSI ratings ranged from 10 to 24 in 1998 at Champaign. In 1999, however, there was a significant ($P \leqslant 0.05$) cultivar by weed management treatment interaction for DSI. The cultivar Jack had a significantly ($P \leqslant 0.05$) greater DSI when growing in plots treated with pendimethalin (DSI = 42), acifluorfen (DSI = 39), or imazethapyr (DSI = 45) compared to the handweeded control (DSI = 12) (Table 1). Weed management treatments or cultivars did not affect severity of Rhizoctonia root and hypocotyl rot at Urbana in 1998, and mean DSI ranged from 4 to 11 (data not shown); however, there was a significant ($P \leqslant 0.05$) cultivar by weed management treatment interaction in 1999. The cultivar Asgrow 3704 had a greater DSI when growing in plots treated with dimethenamid + metribuzin (DSI = 34) when compared to the handweeded control (DSI = 3), and the cultivar Pioneer 9363 had a greater DSI when treated with imazethapyr (DSI = 74) when compared to the handweeded control (DSI = 23).

3.2. Field study 2

Weed management treatments or cultivars did not affect disease severity at Monmouth in 1999, and mean DSI ranged from 36 to 43 (data not shown). In 2000, cultivars did not affect DSI, but weed management treatments significantly ($P \leqslant 0.05$) affected DSI. When imazethapyr was applied, the DSI was 25, which was significantly greater than the glyphosate-only control, which had a DSI of 11 (Table 2).

3.3. Field study 3

Weed management treatments did not have an effect on disease severity at Dekalb in 1999, with mean DSI ranging from 17 to 27 (data not shown); however, weed management treatments did significantly ($P \leqslant 0.05$) affect DSI in 2000. The DSI was increased compared to the glyphosate-only control when acifluorfen or imazethapyr were applied (Table 2).

3.4. Greenhouse PRE herbicide study

There was not a significant ($P \leqslant 0.05$) trial by treatment interaction, so data from both trials were pooled and analyzed together. There was a significant ($P \leqslant 0.05$) cultivar by herbicide interaction for disease severity. When the cultivar Jack was grown in soil treated with pendimethalin or dimethenamid + metribuzin, the disease severity was increased compared to the no-herbicide control (Table 3); however, when the cultivar Savoy was grown in pots treated with pendimethalin, the disease severity did not significantly differ from the no-herbicide control. The herbicide dimethenamid + metribuzin caused both *R. solani* inoculated and non-inoculated Savoy plants to die. This was due to Savoy being extremely sensitive to the herbicide metribuzin (C.D. Nickell, University of Illinois soybean breeder, personal communication).

3.5. Greenhouse POST herbicide study

There was not a significant ($P \leqslant 0.05$) trial by treatment interaction, so data from both trials were pooled and analyzed together. There was a significant ($P \leqslant 0.05$) cultivar by herbicide interaction for disease severity. The cultivar Jack had a greater disease severity rating when treated with imazethapyr at the $2x$ rate compared to the no-herbicide control (Table 3). The cultivar Savoy had a greater disease severity rating when treated with acifluorfen at the $1x$ or $2x$ rate or with

Table 1
The effect of different weed management treatments on Rhizoctonia root and hypocotyl rot disease severity index on six soybean cultivars at Champaign and Urbana, IL in 1999

Cultivar	Weed management	DSI[a]	
		Champaign	Urbana
Asgrow 3704	Handweed[b]	8	3
	Dimethenamid + metribuzin[c]	19	34
	Pendimethalin[d]	3	9
	Acifluorfen[e]	20	16
	Imazethapyr[f]	21	32
Asgrow 3904	Handweed[b]	2	3
	Dimethenamid + metribuzin[c]	3	10
	Pendimethalin[d]	8	6
	Acifluorfen[e]	1	22
	Imazethapyr[f]	3	17
Pioneer 93B65	Handweed[b]	20	20
	Dimethenamid + metribuzin[c]	4	6
	Pendimethalin[d]	2	17
	Acifluorfen[e]	11	11
	Imazethapyr[f]	12	21
Pioneer 9363	Handweed[b]	16	23
	Dimethenamid + metribuzin[c]	16	19
	Pendimethalin[d]	10	28
	Acifluorfen[e]	8	41
	Imazethapyr[f]	20	74
Jack	Handweed[b]	12	53
	Dimethenamid + metribuzin[c]	12	30
	Pendimethalin[d]	42	34
	Acifluorfen[e]	39	34
	Imazethapyr[f]	45	56
Savoy	Handweed[b]	7	11
	Dimethenamid + metribuzin[c]	4	8
	Pendimethalin[d]	5	3
	Acifluorfen[e]	2	4
	Imazethapyr[f]	13	27
	$LSD_{0.05}$[g]	19	24

[a] Disease severity index (DSI) of soybean at approximately V6 growth stage was calculated by (percentage incidence × mean severity)/5, where severity was rated on a 0–5 scale.
[b] No-herbicide handweeded control.
[c] Dimethenamid + metribuzin was applied at PRE at 1.47 and 0.42 kg a.i./ha, respectively.
[d] Pendimethalin was applied PRE at 1.39 kg a.i./ha.
[e] Acifluorfen was applied POST at 0.42 kg a.i./ha + 1% v/v COC.
[f] Imazethapyr was applied POST at 0.07 kg a.i./ha + 1% v/v COC.
[g] Fisher's protected LSD was used to compare means at $P = 0.05$.

imazethapyr at the 1x or 2x rate compared to the no-herbicide control.

4. Discussion

These studies showed that pendimethalin, depending on year, cultivar, and location, could increase the DSI of Rhizoctonia root and hypocotyl rot in the field, and depending on cultivar, it increased disease severity in the greenhouse. These findings support Sumner and Dowler (1983) and Heydari and Misaghi (1998), in which root disease severity and/or damping-off caused by *R. solani* was increased with the application of pendimethalin in corn and cotton (*Gossypium hirsutum* L.), respectively. Bauske and Kirby (1992), however, reported that pendimethalin and other dinitroaniline (DNA) herbicides did not cause an increase in disease caused by *R. solani* based on one field location and one soybean cultivar. There have been several reports of the DNA herbicide, trifluralin, increasing disease caused by *R. solani* on cotton (Pinkard and Standifer, 1966; Chandler and Santelmann, 1968; Neubauer and Avizohar-Hershenson, 1973). Wrona et al. (1981) working with *Phaseolus vulgaris* L., found that trifluralin treated plants had increased size and number of hypocotyl lesions caused by *R. solani*, and attributed it to underdeveloped trichomes on the trifluralin treated plants. More research needs to be conducted to determine if pendimethalin affects trichomes, and what environmental conditions influence this interaction between herbicide and disease.

It was also shown by our results that imazethapyr could cause an increase in severity of Rhizoctonia root and hypocotyl rot. This is similar to reports involving the effect of imazethapyr on sudden death syndrome (SDS) of soybean, caused by *Fusarium solani* f. sp. *glycines*, that showed severity of SDS increased after applying imazethapyr (Sanogo et al., 2000, 2001). Imazethapyr is an acetolactate synthase (ALS) inhibitor. Another ALS inhibiting herbicide, chlorsulfuron, was

Table 2
The effect of different weed management treatments on severity of Rhizoctonia root and hypocotyl rot of soybean at Monmouth and Dekalb, IL in 2000

Weed management	DSI[a]	
	Monmouth	Dekalb
Glyphosate-only control[b]	11	39
Acifluorfen[c]	19	47
Imazethapyr[d]	25	46
Pendimethalin[e]	14	n/a[f]
$LSD_{0.05}$[g]	9	6

[a] DSI of soybean at approximately V6 growth stage was calculated by (pecentage incidence × mean severity)/5, where severity was rated on a 0–5 scale.
[b] Glyphosate was applied at early POST over the entire experiment at 1.12 kg a.i./ha.
[c] Acifluorfen was applied POST at 0.42 kg a.i./ha + 1% v/v COC.
[d] Imazethapyr was applied POST at 0.07 kg a.i./ha + 1% v/v COC.
[e] Pendimethalin was applied PRE at 1.39 kg a.i./ha.
[f] Not applicable.
[g] Fisher's protected LSD was used to compare means at $P = 0.05$.

Table 3
The effect of herbicides on Rhizoctonia root and hypocotyl rot in the greenhouse

Cultivar	Herbicide[a]	Disease severity[b]
PRE herbicide study		
Jack	None	2.7b[c]
	Pendimethalin	4.2a
	Dimethenamid + metribuzin	4.0a
Savoy	None	2.2b
	Pendimethalin	2.7b
	Dimethenamid + metribuzin	n/a[d]
POST herbicide study		
Jack	None	3.7bc
	Acifluorfen 1*x*	4.3ab
	Acifluorfen 2*x*	4.3ab
	Imazethapyr 1*x*	4.3ab
	Imazethapyr 2*x*	4.7a
Savoy	None	3.3c
	Acifluorfen 1*x*	5.0a
	Acifluorfen 2*x*	4.7a
	Imazethapyr 1*x*	4.3ab
	Imazethapyr 2*x*	4.7a

[a] Herbicide treatments for the PRE herbicide study consisted of a no-herbicide control, pendimethalin at 1.39 kg a.i./ha, and dimethenamid + metribuzin at 0.74 and 0.21 kg a.i./ha, respectively. For the POST herbicide study, herbicide treatments consisted of a no-herbicide control, acifluorfen at a 1*x* and a 2*x* rate where *x* was equal to 0.42 kg a.i./ha, and imazethapyr at 1*x* and a 2*x* rate where *x* was equal to 0.07 kg a.i./ha. All herbicides were applied in an automated spray chamber with an 80015EVS nozzle calibrated to deliver 187 l/ha.

[b] Rhizoctonia root and hypocotyl severity rating, where 0 = no lesions, 1 = lesions < 2.5 mm, 2 = lesions 2.5–5.0 mm, 3 = lesions > 5.0 mm, 4 = lesions girdling plant and wilting visible on leaves, and 5 = seedling damped-off or dead.

[c] Means followed by the same letter within a study are not significantly different at $P = 0.05$ using PDIFF to separate least square means in SAS.

[d] Not available, because all Savoy seedlings were killed by the dimethenamid + metribuzin treatment due to sensitivity to metribuzin.

reported to increase disease severity caused by *R. solani* on wheat (*Triticum aestivum* L.) and barley (*Hordeum vulgare* L.) (Rovira and McDonald, 1986; Smiley and Wilkins, 1992).

Our research also showed that the herbicide acifluorfen was able to increase disease severity caused by *R. solani*. It is unclear how acifluorfen and imazethapyr affect *R. solani*, but most likely it is indirect. Since acifluorfen and imazethapyr are POST applied herbicides, very small amounts of the herbicides would come into direct contact with the pathogen in the soil. Acifluorfen has been reported as inhibiting mycelial growth of *R. solani* in vitro (Black et al., 1996), and most other herbicides tested have decreased rather than increased growth of *R. solani* in vitro (Rodriguez-Kabana et al., 1966; Black et al., 1996). The herbicides most likely stress the soybean plant, which predisposes it to infection, similar to what was reported by Canaday et al. (1986) in which herbicide-induced stress increased soybean root colonization by *Macrophomina phaseolina* (Tassi) Goid., the charcoal rot fungus.

The cultivars Jack and Savoy were used in Field study 1 and the greenhouse studies. The cultivar Jack has been reported as being susceptible to *R. solani*, whereas, the cultivar Savoy has been reported to have partial resistance (Bradley et al., 2001). The results of Field study 1 in 1999 agree with this report in that the overall mean DSI of Jack is greater than the overall mean DSI of Savoy at both the Champaign and Urbana locations. The greenhouse PRE herbicide study also agrees with this, in that the overall mean disease severity of Jack is greater than the overall mean disease severity of Savoy. Results from the POST herbicide study, however, do not agree. In the POST herbicide study, the soybean plants were inoculated with *R. solani* at the V1 (first trifoliolate leaf) growth stage compared to the VE (emergence) growth stage, which is when the plants were inoculated in the PRE herbicide study and in that reported by Bradley et al. (2001). It is possible that the cultivar Savoy may lose some resistance as the plants age.

From our data, it was difficult to determine what factors were needed for herbicides to influence disease in the field. Results varied from year to year in the field studies, and effects of herbicides on disease severity were inconsistent and occurred at a low frequency. In the greenhouse studies, the effect of herbicides on disease severity was very clear. The conditions in the greenhouse such as high soil moisture and temperature (>26°C) were conducive for soybean infection and growth of *R. solani* (Lewis and Papavizas, 1977; Liu and Sinclair, 1991). Additionally, inoculum was placed uniformly on and around every plant in the greenhouse. In a field situation, there is not a uniform distribution of inoculum throughout the field, making the accuracy of disease assessments difficult when rating only a small number of plants per plot. The potting mixture used in the greenhouse had a relatively large sand content compared to the soil in the field studies. This also may have had an effect on disease. There were some weather patterns that might have influenced infection of soybean with *R. solani*. At the Champaign–Urbana area in 1999 ≈17–26 days after planting, soil temperature reached nearly 27°C, and rainfall the previous week provided good soil moisture (Fig. 1). According to Lewis and Papavizas (1977) and Liu and Sinclair (1991) the soil temperature and moisture conditions would have been conducive for soybean infection by *R. solani* during that time period at Champaign and Urbana. This may have been part of the reason why differences among herbicide treatments were found in 1999, but not in 1998 at Champaign and Urbana. There was less moisture at Dekalb and Monmouth than at the Champaign–Urbana area, but there is little indication as to why disease was

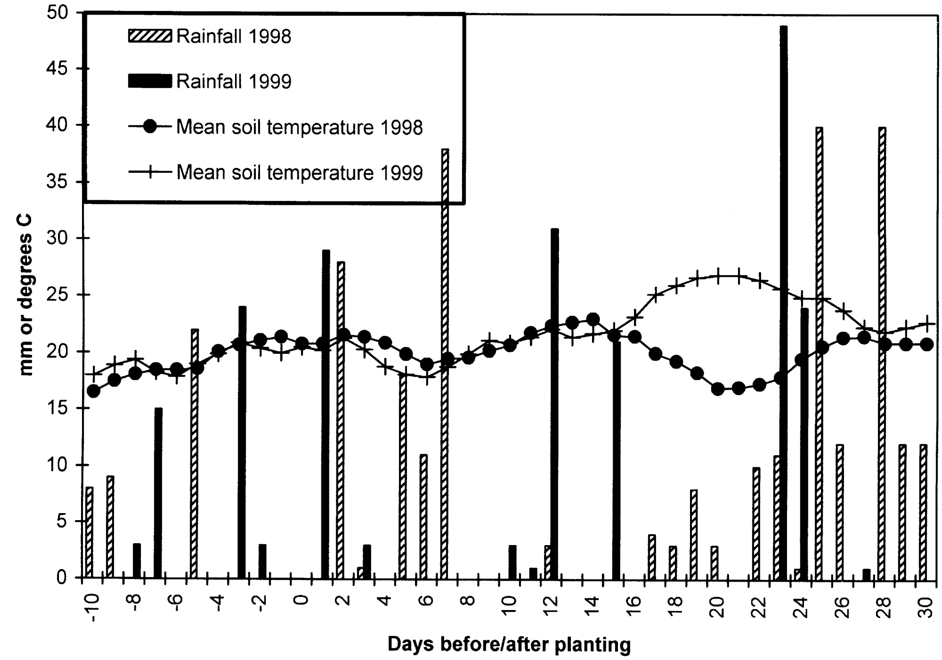

Fig. 1. Rainfall and mean soil temperature at a depth of 10 cm for the Champaign–Urbana, IL area in 1998 and 1999.

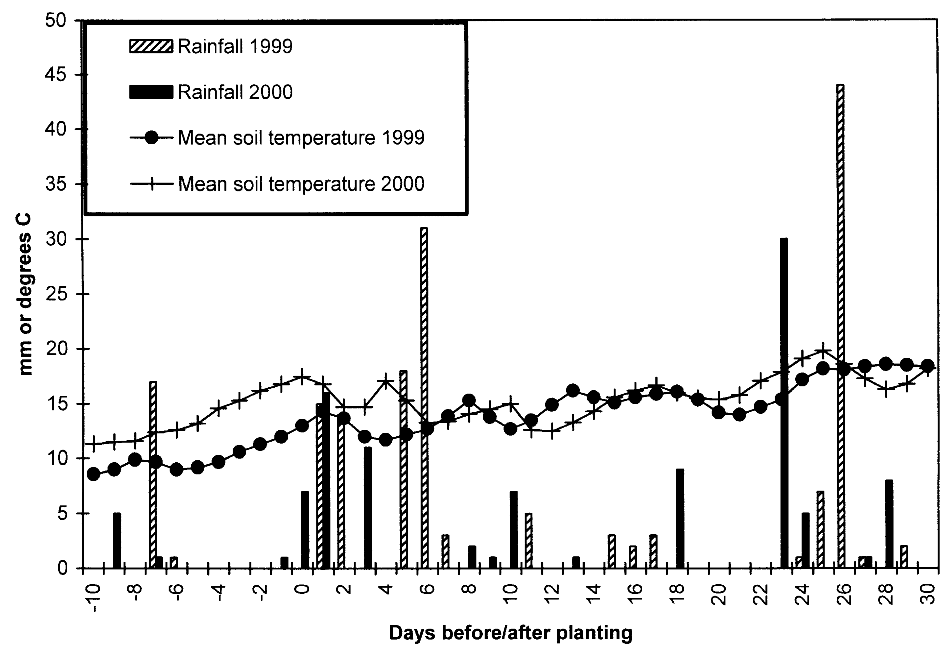

Fig. 2. Rainfall and mean soil temperature at a depth of 10 cm for Dekalb, IL in 1999 and 2000.

affected by herbicides at Dekalb and Monmouth in 2000, but not in 1999 (Figs. 2 and 3).

In our studies, the increases in disease severity associated with herbicides were statistically significant, but they were variable among locations, cultivars, and years, and were not consistent within a particular treatment. The economic importance of damage to soybean caused by *R. solani* is not fully understood. Yield reductions associated with Rhizoctonia root and hypocotyl rot of soybean are difficult to assess, and soybean plants are able to compensate well for missing plants that have damped-off at an early stage (Stivers and Swearingin, 1980). More research is being done to determine the environmental conditions necessary for

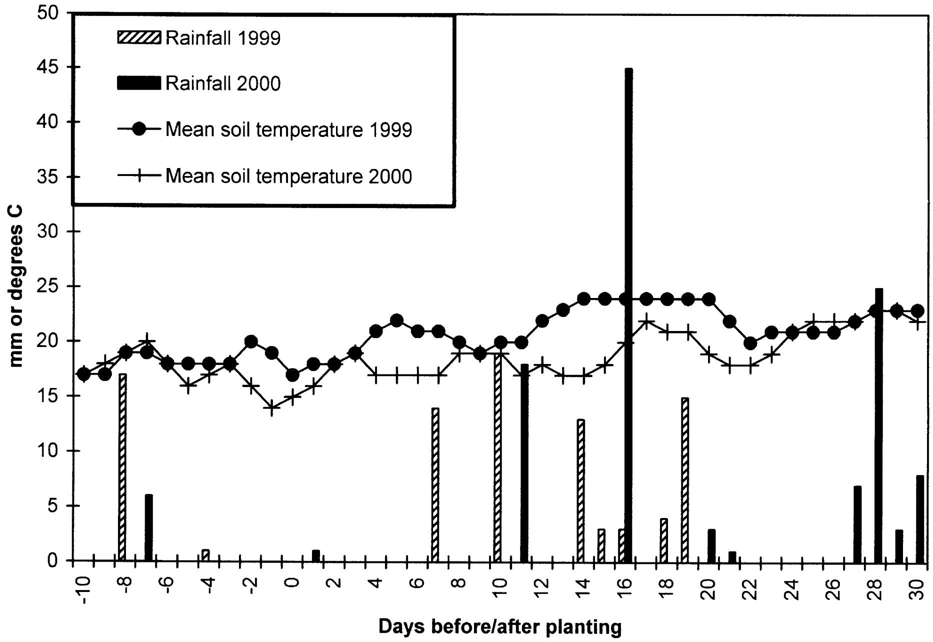

Fig. 3. Rainfall and mean soil temperature at a depth of 10 cm for Monmouth, IL in 1999 and 2000.

herbicides to impact *R. solani* disease severity and soybean growth and yield, and the economic importance of *R. solani* to soybean.

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